RY22

CLINICAL CASE STUDIES

Dietary Intervention in Systemic Lupus Erythematosis:

4 Cases of Clinical Remission and Reversal of Abnormal Pathology

† *Cooke, Helen M., R.N.*
12 Kidston Crescent
Curtin, ACT 2605
Australia

Reading, Chris M., B.Sc. Dip. Agr. Sci., MB, B.S., MRANZCP
Consultant Psychiatrist
Pacific Medical Centre
Dee Why, N.S.W. 2221 Australia

Introduction

In their summary of 25 years treatment of Systemic Lupus Erythematosis (SLE), Morrow and colleagues[1] highlighted the attractions of possible dietary treatment, with its low risk of side effects. They discussed findings involving dietary restriction of fats, calories and zinc, all of which have been shown to influence the course of murine lupus.

Our observations suggest other aspects of dietary treatment worthy of investigation. Case reports are presented indicating remission in 4 SLE patients from one Australian psychiatric practice. These patients are undergoing treatment for nutritional/allergy/sensitivity problems and related psychiatric symptomatology. Prolonged remission of abnormal pathology has resulted, together with apparent clinical improvement. Degree of improvement was noted by our consultant psychiatrist, the patient and each patient's independent referring doctor. Other relevant information obtained retrospectively from each patient's and her referring doctor's questionnaires is also included.

Our belief that the reported remissions are directly related to the dietary intervention, rather than to spontaneous remission common to SLE, is strengthened not only by observations of a similar trend in another 70 patients with lupus and lupus-like syndromes, but also by repeated observations of an intriguing association between many varied apparently lupus-related symptoms and exposure to various foods, chemicals and inhalants.

Recent findings reported in the Allergy Special Supplement to the Medical Journal of Australia[2] support the existence of adverse food and chemical reactions resulting in delayed and multiple organ symptoms, though mechanisms remain unclear. Allen and co-workers, who have documented systemic reactions by repeated double-blind challenge in 74 patients, recognise that the vast majority of adult food and chemical reactions are 'delayed' rather than 'immediate', and are mediated by other than IgE related mechanisms. They have found neurological symptoms to be very frequent in systemic reactions, often most bizarre, and mimicking those of neurosis and organic brain syndrome.

Apparent involvement in 'delayed' reactions[3,4,5,6] of leucotrienes prostaglandins, complement, histamine, as well as other mediators and immunoglobulins is particularly interesting in relation to our findings, as abnormalities involving most of these substances have been shown to exist in SLE, or have been shown to affect the course of the disease.

† *AUTHOR FOR CORRESPONDENCE AND REPRINTS*

CASE REPORTS

Case 1

This 41 year old woman, who reported lupus-like symptoms since age 15, complained of alopecia, photosensitivity, mucosal ulceration, muscle pain, gastrointestinal symptoms, severe fatigue, and psychiatric symptoms including depression, for which she had been hospitalised for 6 months and had been taking 12 Tolvon daily. As a last resort, having been booked in for psychosurgery recommended by 4 psychiatrists because of deteriorating depression over a 10 year period, she sought consultation in our practice. SLE was diagnosed following consideration of the clinical picture and laboratory tests showing ANF 1:40, ds DNA 28, and skin biopsy positive for IgM. In the 4 years since that time, treated with 'allergic' avoidance and nutritional supplements, she has avoided psychosurgery and has progressed extremely well, is now off all psychiatric and other medication, and is leading a normal life, even coping with her 6 children with little more than occasional moodiness. ANF and DNA following dietary intervention became negative. The improvement rating of her independent referring practitioner was 'significant'.

Case 2

This 54 year old woman reported lupus-like symptoms since age 8, and at diagnosis complained of photosensitivity, mucosal ulceration, joint and muscle pain, swollen lymph glands, kidney problems, central nervous system (CNS) symptoms, anaemia, Sjogren's syndrome, gastrointestinal symptoms, severe fatigue and fever. Laboratory tests showed ANF 1:640, ds DNA 64 and skin biopsy positive for IgM, IgG, Clq and C3, ESR 5.

The independent referring doctor's improvement rating was 'slight' which contrasted with 'extraordinary — about 70%' as rated by the patient following cessation of 6 symptoms and improvement of others during the 3 years on nutritional treatment. The patient's rating is supported by reversal of ANF 1:640 to completely negative, DNA from 64 to negative and a now negative skin biopsy.

Case 3

This 40 year old woman reports 10 years of lupus-like symptoms before diagnosis, at which time she reported alopecia, mucosal ulceration, CNS symptoms, anaemia, bruising and gastrointestinal symptoms. Laboratory tests showed ANF 1:60, negative DNA, but skin biopsy positive for C3, ESR 3. On nutritional treatment, her ANF became negative and her clinical improvement was rated by her independent referring doctor as 'significant'. The patient reported relief from alopecia, anaemia and bruising as well as improvement in other symptoms increasing during the 18 months of treatment. Her questionnaire included an added comment that she now felt well for the first time in many years.

Case 4

This 35 year old woman had a two year history of intermittent skin rashes, photosensitivity, mucosal ulceration, joint pain, swollen lymph glands, headache, depression, irritability, increasing infections, spontaneous bruising, muscle pain and spasm, gastrointestinal symptoms, and has since developed paraesthesia of right arm, neck and face. At SLE diagnosis ANF was 1:320, ds DNA 36, C4 and CH50 low, IgM raised, anti-lymphocyte antibodies 1:2, and skin biopsy positive for IgG, IgM, Clq and C3 at dermo-epidermal junction. As improvement on nutritional therapy alone was not rapid enough to allow her to cope with her significant family problems, she was also given Decortysil 5-15mgms for 8 weeks, resulting in clinical improvement. Four months after ceasing cortisone, clinical improvement continued with ANF 1:500 which gradually reversed to negative within twelve months, accompanied also by return of DNA and IgM to normal. In the four years on nutritional therapy, two attempts to greatly relax the dietary regime resulted in detiorating general health, increased lupus-like symptoms, and abnormal pathology (ANF) which was reversed again to negative, without steroids, when the dietary regime was recommenced. Clinical improvement was rated by this patient's independent referring doctor as 'significant'.

This patient's complete absence of symptoms when avoiding substances to which she reacts, and claims of prompt re-emergence of presumably lupus-related symptoms, e.g. headache, fatigue, irritability, depression, joint and extensive muscle pain, paresthesia and spontaneous bruising, on exposure to particular substances, prompted her hospitalisation under another independent physician in a specially constructed allergy unit where chemical and food exposure could be controlled and consequences of challenge noted. Fasting for 5 days in this unit on bottled spring water only, resulted in intensification of symptoms (joint and muscle pain, headache, bruising and depression) for the first 2-3 days, then gradual improvement to completely symptom free baseline for testing which began on the 6th day. Open challenge during the following 3 weeks produced no reaction to some foods, generally those not eaten regularly. However, similar single food challenges to numerous other foods, were followed by re-emergence of symptoms in varying combinations. 'Positive' foods included most protein sources, most grains, some fruits and some vegetables. Some symptoms (blurring vision, burning and tingling sensations of right arm, neck and face, rapid pulse, flushing weakness and dizziness with inability to stand or walk) occurred promptly within 5-30 minutes. Other symptoms (nightmares, feelings of 'impending doom', headache, muscle aching) occurred 4-18 hours later, frequently at night, whereas some joint pain appeared not to manifest until 36-48 hours after exposure.

Similar reactions followed open and patient-blind test exposures to fumes or contact with numerous environmental chemicals, e.g. car exhaust, diesel, gas, formaldehyde, chlorine, tobacco, newsprint, synthetic carpet, rubber and polyurethane. Patient-blind challenge using extracts of foods and chemicals sublingually and intradermally has since confirmed symptom provocation with over 15 previously tested substances.

Following significant reduction of exposure to environmental chemicals, particularly in her home, this woman has resumed a full, active life and has taken up 'jogging'. Within 6 months of leaving the hospital, she ran a slow but steady non-stop 10k 'fun-run', with no ill effects.

OUTLINE OF NUTRITIONAL REGIME

Following referral to this practice, patients with a puzzling assortment of psychiatric and/or physical complaints, are asked to assist in compiling a medical family tree. Information can usually be collected for four generations and takes into account illnesses and relevant signs and symptoms suggesting subclinical or undiagnosed disease. Very frequently this information reveals obvious increased clustering and combinations of conditions such as psychiatric illness, allergies, behaviour problems, learning difficulties, coeliac disease, other gastrointestinal complaints particularly I.B.S., diabetes, asthma, arthritis, migraine, pernicious anaemia, other autoimmune disease, cardiovascular disease and cancers.

Closer examination, making use of genetic markers such as congenital abnormalities e.g. colour blindness, as well as distinctive physical features e..g. stature. eye, skin or hair colour, often reveals likely or clear-cut patterns of genetic transmission. Of particular relevance in SLE appears to be the presence of underlying X-linked food allergies/sensitivities with consequent vitamin and mineral deficiencies. These appear to be the common thread for many of the previously mentioned conditions in these families, with many such conditions unexpectedly responding to dietary elimination and vitamin and mineral supplementation[7].

Comparison of features of milk/grain allergies, coeliac disease, and SLE reveals many similarities[8]. Immunofluorescent techniques are now confirming the presence of antibodies to various fractions of milk, grain, egg and yeast products (table 2), which appear to be the immunogenic components of these foods, in susceptible people[8].

Actual foods best avoided in SLE, and those considered safest in our experience, are detailed in Tables 1 and 2.

In order to identify specific deficiencies and imbalances resulting from chronic but often subclinical G.I. inflammation, patients whose family tree or other history supports the likelihood of food allergy/sensitivity, are now tested for vitamin and mineral deficiencies and imbalances, as well as for food allergy by R.A.S.T. and cytotoxic tests. As they have become available, food fraction tests have been included. Full blood count, multichannel analysis (SMA), thyroid tests and tests for autoantibodies, ANA, DNA, complement, immunoglobulin profile, protein electrophoretogram and skin biopsy are also carried out if indicated.

Table 1

SAFEST FOODS FOR SLE PATIENTS
(provided not allergic/sensitive following R.A.S.T., Cytotoxic, provocative or challenge tests).

GRAINS etc.	Rice, sunflower seed, sesame seed, lentils/legumes. For bread, pastry — rice flour, arrowroot flour, potato flour, sweet potato flour, soya flour, lentil flour, chick pea flour, tapioca flour, sago flour.
MILKS, and SUBSTITUTES etc.	Goat's Milk (not cheese or yoghurt). For a baby, human milk with mother off cow's milk/gluten and other food allergies). Coconut milk. Agar instead of gelatin.
MEATS, FISH etc.	Chicken (unseasoned), turkey, duck, goose, pheasant, lamb, pork, rabbit, goat, deer. Fish, especially sardines, pilchards, whiting, mackerel, salmon, bream, jewfish, kingfish, flounder, tuna, trout, snapper, flathead, etc., oysters, crab, squid, calamare (octopus), lobster, prawns, scallops.
NUTS	Cashews, almonds, hazelnuts, macadamia, brazilnuts, pecans, coconut.
VEGETABLES	Tomato (twice weekly), potato, sweet potato, strawberries, peas, string beans, soy bean, lentils, cabbage, cauliflower, brocolli, brusellsprouts, dill, carrots, pumpkin, celery, cucumber, (occasionally) spinach, watermelon, raddish, watercress, parsnips, lettuce, sugarbeet, citrus, rhubarb.
FRUITS, BERRIES	Citrus occasionally — oranges, lemons, grapefruit, mandarines, tangarines, limes. Grapes (washed), raspberries. blackberries, mulberries, loganberries, gooseberries, red currants, avocado (occas.), pineapple, apricots, peaches, persimmon, lychees, blackcurrants, pawpaw, mango, apple (peeled) pear, custard apple, quince, passionfruit, kiwifruit, big bananas. plums/prunes, figs (occas.), dates (occas.).
MISCELLAN.	Tea, maplesyrup, coffee, honey, carob, parsley, safflower.

Cytotoxic test results have served as useful pointers to other problem foods, many of which would not have otherwise been suspected[9]. Cytotoxic positive foods whose relevance is confirmed by symptom provocation on challenge after avoidance, are further avoided, but can sometimes be reintroduced in moderation after several months avoidance.

For the four patients here reported, individual dietary regimes were suggested, depending on medical and family history, results of R.A.S.T. food fraction and cytotoxic tests, and degree of response to initial trial elimination of dairy, egg, yeast and gluten containing products. Patients were also advised to avoid artificial colours, flavours, herbs, spices and preservatives as well as highly refined foods, and minimise sugar intake. Other environmental chemicals were not excluded initially, but individual patients have reported benefits from consideration of this aspect.

Table 2.

FOODS TO BE AVOIDED FOR DIETARY INTERVENTION IN SLE.

The following foods (and pollens as far as possible) are best avoided for at least 18 months — some indefinitely as symptoms of SLE keep returning when those foods are re-introduced.

GRAINS	DAIRY PRODUCTS	EGGS	YEAST
Wheat, Rye, Oats, Corn/Maize, Malt Millet, Barley Buckwheat Cane sugar/syrup Grass pollens Tree pollens	Cow's milk, Cream, Cheese, Butter Yoghurt, etc. Products containing Casein. Beef Gelatine		Both Baker's yeast and Brewer's yeast, and yeast fractions as in wine, vinegar, Bread, mushrooms, wallnuts, and moulds.
Gluten and gliadin and other glyco-protein fractions in wheat/grains. Glycoprotein fractions in sugar, and glyco-proteins in pollens of grasses and trees.	DAMAGING FRACTIONS. Especially casein, lactalbumin, and lactoglobulin, and beef albumin and globulin.	Ovalbumin and egg globulins.	Glycoprotein fractions of yeast moulds and virus capsules. (Cf. GP 70 in capsule in Type cRNA viruses in murine SLE.

Other Foods Contraindicated in SLE

Curry, chili, sauces, spices, herbs, seasonings (contains pressor amines and vasoactive peptides, that dilate capillaries, increasing permeability of toxic fractions, etc.)

Food additives, colourings, preservatives (as well as certain drugs, oestrogens, offending petrochemicals, etc., and too much sunlight).

Foods also frequently observed to aggravate SLE (in over 90 cases)

Onions, garlic, asparagus, ginger, peppers, capsicums, egg plant, paprika, choko, zuchini, mustard, small bananas, olives, chocolate, peanuts, walnuts, pistacchio, cinnamon, nutmeg, cola, kidney beans, mung beans, licorice, oregano, sage, cloves, poppy seeds **and specific food allergies/intolerances.**

Foods to be limited to 2-3 times weekly — *citrus, *tomatoes, cucumber, avocado.
*Totally avoid if antibodies to R.A. Latex, Rose Waaler or Synovial Membrane present.

All patients were administered nutrition supplements with dosages varying depending on knowledge available at the time, experience with previous patients, results of serum estimations, clinical progress and/or patients' tolerance. Preparations were recommended according to bioavailability and content, taking into account other constituents e.g. nature of fillers, binders, etc. to avoid potentially allergenic substances (e.g. yeast in B vitamins, wheat germ or oil, lactose, corn or wheat starch, etc.).

Actual supplements and dosage ranges taken by the 4 patients here reported are given in Table 3.

Table 3

RECOMMENDED SUPPLEMENTATION FOR SLE PATIENTS

Supplement	Daily Intake-Range	Number of Patients Supplemented
Vitamin A	9,000 daily for 1 month, or alternate months	2
B_1	100-300mgms	4
B_3	500-1000mgms	2
B_6	250-750mgms	4
Multi-B	1-2 capsules	4
Multivitamin	1 capsule	4
Vitamin C	2000-5000mgms	4
Vitamin E	250-500IU	4
Calcium	400-800mgms	4
Magnesium	200-400mgms	4
Zinc	25-75mgms	4

Other
B_2 — 100mgms (1 patient)
B_{12} — 1000mcgm injection 2nd weekly (1 patient)
Intravite — 2ml weekly (1 patient)
Folic acid — 5mgm (1 patient)
B_5 — 100mgm (1 patient)
Adrenal tissue extract — 4 tabs (1 patient)
Thymus tissue extract — 2 tabs (1 patient)

Other Outcomes and Considerations

Dependence on psychiatric medication by these patients has been reduced, with no patient still requiring any regular psychiatric medication. Other than recent hospitalisation of patient 4 for investigation of chemical and food sensitivities in an allergy unit, no patient has required hospitalisation since commencing nutritional treatment. This covers a period on treatment averaging 28 months, and varying from 18 months to 4 years, and contrasts with a total of 22 weeks prior hospitalisation by 2 patients for SLE related symptoms.

Evaluation by an independent dietition, of probable dietary adequacy based on brief retrospective questionnaire information, suggested that all of the four patients appeared to have been consuming nutritionally adequate diets at diagnosis as judged by representation of food groups. Retrospective nature of dietary information and length of time involved, prevents more accurate assessments.

Serum vitamin levels were tested in two of the 4 patients, both of whom registered abnormal levels (usually low), despite apparently adequate diet and in one patient (patient 4) despite 6 months supplementation.

Future studies should obtain detailed current nutritional analysis and history before treatment in order to begin to clarify relationship of nutritional inadequacies to intake, poor absorption or metabolic aberrations. This would also help to determine and document the dosage of supplements needed to restore normal values and functions. In our experience very much higher levels than RDA appear to be necessary.

Side Effects

Other than withdrawal symptoms and temporary relatively minor sensitivity symptoms from non-tolerated supplements, no side effects have been reported by referring doctors or patients in separate retrospective questionnaires, except patient 3 who reported initial loss of stamina.

Discussion

Nevertheless, high doses of anything including vitamins, must be used with caution, and likely side effects weighed up against observed benefit, as in any other medical treatment. Our preliminary observations suggest that this approach compares more than favourably with usual lupus treatments which not uncommonly involve significant and potentially dangerous side effects. The most likely danger appears to relate to possible dietary inadequacy or nutritional imbalance caused by multiplicity of food sensitivities, inadequate supervision and/or ignorance of possible food substitution. Competent encouraging advice from qualified dietitians would be valuable but has not been generally available.

Two opposing views on uselessness or justification for vitamin supplementation are represented by articles by Herbert[10,11] and Bland[12]. The few studies reporting harmful effects of high dose supplementation have related to inadequately or medically unsupervised use of fat soluble vitamin A and E, as well as water soluble B_6, but all in massive amounts — far greater than those used for these patients. A 50 page supplement of the American Journal of Clinical Nutirition[13] titled 'Single Nutrients and Immunity' presents strengths and weaknesses of available data, and demonstrates the rapidly growing scientific interest in, and knowledge of nutrition in health and disease. It becomes apparent that, despite problems in design, execution or interpretation of some studies, evidence is accumulating to demonstrate unique and profound effects of single and multiple nutrients on immune competence.

The possibility exists that the patients here described may be a subgroup with less severe illness as described by Gossatt and Walls[14]. This is supported by the average age at diagnosis being 42 years, and the lack of significant renal complications, but argued against by the unexposed skin biopsy finding of IgG in 2 of the 4 patients biopsied — a finding usually associated with more severe disease and poorer prognosis.

That SLE patients may have multiple allergies does seem likely, considering the not infrequent co-existence in SLE of low S-IgA[15,16] and the generalised rather than specific nature of B cell hyperactivity[17,18,19]. Drug and chemical allergy reactions are known to occur frequently in SLE[20] and patients are advised to avoid drugs to which they have had adverse reactions, to prevent 'flares' of disease activity. Curiously other everyday aspects which may combine to create much greater exposure, are largely ignored, in spite of reports of increased prevalence and higher titres of antibodies to numerous food fractions in SLE patients' serum[21].

Evaluation of allergic status in SLE has revealed conflicting conclusions, with anergy, rather than allergy in some patients. Where allergy is obviously present, it is presumed to be secondary to other immune aberrations, perhaps because some patients have a positive allergy history before diagnosis, but others do not.[22,17,18]. However, the possibility of masked non-atopic allergy/sensitivity, or sudden development of immune dysregulation and hyperactivity following severe stress or chemical overexposure, has not been excluded, and should be sought. As is obvious from the rapidly growing knowledge of delayed adverse reactions[2,3,4,5,6] it is no longer valid to consider adequate 'allergic' assessment to be based on simple skin or blood tests capable of identifying only IgE mediated reactions.

Two other possibilities contributing to reports of anergy in SLE are:—

1. Anergy may result as immune suppression follows earlier immuno-enhancement and extends to bone marrow cells[5,17]. In animals, severe depression of T-suppressor cells results in low allergic reactivity, yet less severe depression results in immune enhancement[5]. Those patients showing anergy may have been very ill with advanced active disease.

2. Antigenic competition. It is suggested that depression in cellular immune response in SLE may be associated with antigenic competition[17]. Experimentally, reactions to a test antigen can be reduced or negated by previous or simultaneous exposure to another or other antigens[23]. The intense B cell activity would be expected to result in a form of antigenic competition in which the production of autoantibodies would lead to the production of non-specific suppressor factors, which would inhibit the primary immune response[17]. Perhaps this helps explain difficulties in some patients with significant, though unsuspected chemical sensitivity. Even elimination diets may fail to identify and confirm concommitant food sensitivities until major chemical factors are removed, thus necessitating diagnostic testing in allergy units[24] as described by Dickey[25] and Rea[26,27]

Future Directions

For some SLE patients sensitive to a wide range and number of foods and chemicals, particularly those unwilling or unable to avoid foods to which they react, the cure by avoidance, may seem as bad as the condition. Most people welcome the chance to regain control of their health, and cope with the increased workload in the kitchen, and disruption to their social life, with determination and optimism. Others find the benefits of food avoidance outweighed by the stress of dietary and other lifestyle restrictions, and wish they had never heard of this approach. Still others are sensitive to so many foods that it may seem impossible to ensure adequate nutrition; some even become sensitive to the foods to which they are restricted, and do not achieve sufficient benefit to make up for the extra effort and perceived deprivation. Children, particularly teenagers, often don't comply with the necessary restrictions, and this can have serious repercussions with family relationships.

In such situations when management is so difficult, great hope may lie with clinical ecology procedures involving serial dilution titration, provocation/neutralisation techniques and rotation diets. Despite earlier denouncement by eminent colleges, the effectiveness of these techniques is now being reported in acceptable double-blind studies[28,29,30,31,32,33]. It is interesting to note that cognitive and emotional symptoms have been verified scientifically[28], as well as numerous other symptoms considered by many to be of psychological origin (eg. irritable bowel syndrome, enuresis, frequency, insomnia, headache). As well as assisting in identifying food and chemical incitants, use of these techniques provides a treatment consisting of sublingual drops of extracts, which allows consumption of previously reactive foods without, or with minimal, reaction.

With respect to SLE, caution is urged on three counts. One is that there are very few allergists or other medical practitioners who are proficient in these particular techniques. Secondly, the effects of these neutralisation testing and treatment procedures on the

already malfunctioning SLE immune system have not been explored. Thirdly, while avoidance in SLE has been repeatedly observed by us, to be followed by reversal of abnormal pathology, the same cannot yet be said with neutralisation until more patients have been followed. A double-blind crossover single case study which will involve observation of clinical and pathology results during periods of active extract and placebo administration, is about to be undertaken on the first SLE patient so treated (Patient 4 from this paper), and will, if successful, be followed by others, when these patients have also used these techniques for six months with apparent benefit.

The extent of environmental chemical sensitivity in SLE patients is not yet known. Initially, it was not recognised that environmental chemicals could also precipitate symptoms in SLE patients. However, as patients' SLE symptoms and general health improved, and they no longer felt ill most of the time, some reported prompt recurrence of symptoms on exposure to chemicals such as aerosol sprays, perfumes, gas, diesel and exhaust fumes, paint, hair perm solution, new carpet, etc. In such cases patients needed little convincing as to the necessity of avoiding or minimising such exposures. The first two SLE patients to be tested patient blind for chemicals such as phenol and formaldehyde have both responded promptly (within 10 minutes) with symptoms — mainly headache, paraesthesias, nausea and fatigue. Subsequent reduction or cessation of these symptoms with neutralisation techniques has led to the daily administration of sublingual extracts to reduce reactions from these and other largely unavoidable substances. The reported ability of low-dose environmental chemicals, to compromise immune function in animals[33] and reported development of multiple food and chemical sensitivity after chemical overexposues[34] raise the possibility that failure to adapt to environmental chemicals, particularly if exposed to high concentrations, may be a major aetiological factor in at least some SLE patients. It would be interesting to know whether there is a higher than expected incidence per percentage of population of nurses, hairdressers, laboratory staff, or office workers whose jobs include operation of photocopiers for extended periods, amongst SLE patients.

Rotation diets, as outlined by Dickey[25] may result in foods of lesser sensitivity being better tolerated, since any food is only used no more frequently than on one day in four. This also seems to lessen development of further sensitivities, but is only possible if one has a reasonable range of foods to rotate. Even when sensitivities exist to most foods, neutralisation to 4 protein sources, 4 grains, 4 yellow vegetables, 4 green vegetables, 4 fruits and a few miscellaneous foods such as potato, yeast, honey, safflower and sugar, perhaps even tea and coffee, provides the basis for an acceptable, nutritious, though obviously repetitive diet. For some, rotation with neutralisation may only be necessary for a limited period, and could be followed by relaxation of rotation procedures providing SLE pathology remained acceptable.

Below is an example of a 4 day rotation diet used by patient 4 in combination with neutralisation therapy, and includes most common foods. It should be noted that designing a practical acceptable normal-look rotation diet is not easy, as every book or article written in the subject gives different information. In addition, personal preference will influence which foods combine well with what. Sweeteners and spreads, e.g. butter, margarine, are difficult to rotate, because of limited choice or reduced availability of alternatives. Whilst not technically perfect this regime has been used successfully and is given as an example.

Should neutralisation techniques not be available or effective (some patients may not obtain sufficient neutralisation of phenol, the preservative in the extract, or their levels may change frequently depending on other stresses), the above regime may still be useful with obvious modifications. Eliminating gluten-containing grains, dairy products, yeast and egg requires avoidance of rye on Day 1, wheat, barley, milk, yoghurt, cheese, butter and yeast on Day 3, and egg on Day 4. Without these foods, sufficient choices remain, except perhaps for Day 3 where it may be necessary to use rice or tapioca as on Day 1, and eat less common

meat such as venison, duck, prawns, etc., or another type of fish.

Obtaining sufficient calories can be a problem, particularly where some degree of malabsorption persists. This can be overcome by making 'pikelets' using a combination of the flours of the day, and sweetener e.g. honey, mixed with a little oil or margarine (milk-free) and cooked like pikelets in an electric frypan. Many other food sources are available and may be acceptable, particularly if patients are willing to look beyond the staples of their own cultures.

	DAY 1	DAY 2	DAY 3	DAY 4
GRAIN	Oat Rice Rye	Potato Soy	Corn Wheat Barley	Arrowroot Buckwheat
PROTEIN	Pork	Lamb Fish	Beef Cheese Veal	Chicken Egg
YELLOW VEG.	Carrot	Tomato	Corn	Pumpkin
GREEN VEG.	Broccoli	Bean	Silver beet	Pea
FRUIT	Apple Pear Strawberry	Peach Grape Mango	Orange Blackberry	Pineapple Banana Rockmelon
EXTRAS	Cabbage Honey Cocoa Tapioca Coconut Carob	Soy Onion Capsicum Black pepper Safflower Eggplant	Yeast Yoghurt Sugar Milk Malt Rhubarb	Apricot Kiwi fruit Lettuce Parsley Cherries
FAT. OIL	Pork fat	Safflower oil	Butter	Chicken fat

Conclusion

The possibility that nutritional intervention can alleviate some chronic and even acute symptoms as well as improving stress tolerance and quality of life in even a subgroup of SLE patients, warrants intensive investigation. The apparent association between lupus-like symptoms and 'allergic' challenge is intriguing. Whether this is of fundamental aetiological significance in susceptible people, or whether 'allergic' exposure aggravates SLE by secondary effects as an immune stimulant or as yet another stress factor, it does appear logical to attend more closely to the 'allergic' and nutritional status of SLE patients.

Research is needed to establish the mechanisms involved in the reported allergy/sensitivity reactions, and to further document benefits of this and other types of dietary intervention. Current forms of treatment are far from satisfactory and the need for improved forms of treatment is of paramount importance[1], as human SLE is an unpredictable highly variable disease, that remains difficult to monitor and treat.

REFERENCES:

1. Morrow WJW, Isenberg DA, Youinou P, Snaith ML. Systemic lupus erythematosis: 25 years of treatment related to immunopathology. **Lancet** II, 206-210 (1983).
2. Allen DH, Van Nunen S, Loblay R, Clarke L. Swain A. Adverse reactions to foods. **Med J Aust** Sept 1 (suppl): S37-42 (1984)
3. Bryant DH. Allergies, mediators and asthma. **Med J Aust** Sept 1 (suppl):S2-5 (1984)
4. Baldo BA, Tovey ER. Allergy, allergens and

allergen standardisation. **Med J Aust** Sept 1 (suppl):S5-8 (1984)

5. McGovern JJ, Lazaroni JL, Saifer P, Levin AS, Rapp DJ, Gardner RW. Clinical evaluation of the major plasma and cellular measures of immunity. **Orthomolecular Psychiatry 12(1)** 60-71 (1983)
6. Little CH, Stewart AG, Fennessy MR. Platelet serotonin release in rheumatoid arthritis: a study in food intolerant patients. **Lancet II**, 297-99 (1983)
7. Reading CM, Meillion In: Relatively speaking. The family tree way to better health. Sydney, Australia. Fontana (1984)
8. Reading CM. Systemic lupus erythematosis: address to the 13th Annual Conference: the McCarrison Society Sept. 23 (1983) **Nutrition and Mental Health:** The Links between Food and Behaviour (1983)
9. Reading CM. Relevance of cytotoxic test to detect food allergies/intolerance/hypersensitivity in psychiatric patients. Schizophrenia Association of Great Britain Conference, 22 April, 1982 Recent Trends in Biological Psychiatry.
10. Herbert V. The vitamin craze. **Arch Intern Med 140**; 173 (1980)
11. Herbert V. Facts and fictions about megavitamin therapy. **Resident and Staff Physician** Dec: 43-50 (1978)
12. Bland JS. The justification for vitamin supplementation. **Orthomolecular Review 2(2)** 4-13 (1982)
13. Beisel WR. Single nutrients and immunity. **Am J Clin Nutr 35** (suppl); 2, 417-458 (1982)
14. Gossatt DM, Walls RS. Systemic lupus erythematosis in later life. **Med J Aust 1**; 297-9 (1982)
15. Hong R, Ammann AJ. Selective absence of IgA. **Am J Pathol 69(3)**; 491-5 (1972)
16. Ammann AJ, Hong R. Selective IgA deficiency: presentation of 30 cases and a review of the literature. **Medicine 50(3)** 223-36 (1971)
17. Raveche ES, Steinberg AD. Lymphocytes and lymphocyte functions in systemic lupus erythematosis. **Seminars in Haematology 16(4)**; 344-70 (1979)
18. Taurog JD, Steinberg AD. Genetic and immune aspects of systemic lupus erythematosis. **Int J Dermatology 20**; 149-58 (1981)
19. Kumagai S, Sredni B, House S, Steinberg AD, Green I. Defective regulation of B lymphocyte colony formation in patients with systemic lupus erythematosis. **J Immunol 128(1)**; 258-62 (1982)
20. Hughes GRV, Lachmann PJ. Systemic lupus erythematosis. In: Clinical aspects of immunology (3rd ed). Oxford: Blackwell 40; 1136 (1975)
21. Carr RI, Wold RT, Farr RS. Antibodies to bovine gamma globulin (BGG) and the occurrence of a BGG-like substance in systemic lupus erythematosis sera. **J Allergy Clin Immunol 50(1)** 18-30 (1972)
22. Rebhun J, Dubois E. Skin response to histamine in systemic lupus erythematosis. **Ann Allergy 42**; 148-51 (1979)
23. Adler FL. Competition of antigens. **Progr Allergy 8**; 41-57.
24. Felleson JA. The clinical ecology unit. RN 1977; March 49-57.
25. Dickey Ld. (ed). In: Clinical Ecology 1975, Springfield Ill. Thomas.
26. Rea WJ. Environmentally triggered cardiac disease. **Ann Allergy 40** (April) 243-51 (1978)
27. Rea WJ. Environmentally triggered small vessel vasculitis. **Ann Allergy 38** (April) 245-51 (1977)
28. King WJ. Can allergic exposure provoke psychological symptoms? **Biolog Psych 16(1)** 3-19 (1981)
29. Boris M, Schiff M, Weindorf S, Inselman L. Bronchoprovocation blocked by neutralisation therapy. **J Allergy Clin Immunol 71;92** (1983)
30. Miller JB. A double-blind study of food injection therapy — a preliminary report. **Ann Allergy 38**; 185-191 (1977)
31. Lee KL, Kniker WT, Campos T. Aggressive coseasonal immunotherapy in mountain cedar pollen allergy. **Arch Otolaryngology 108**; 787-794 (1982)
32. Forman R. A critique of evaluation studies of sublingual and intracutaneous provocative testing for food allergy. **Med Hypoth 1**; 1019-1020 (1981)
33. La Via: MF, Loose LD, La Via DS, Silberman S. The Immunosuppressive effect of phenol derivatives. **Advances in Exper. Med. Biol. 523**, Plenum (1979)
34. Rea WJ, Bell IR, Suits CW, Smiley RE. Food and chemical susceptibility after environmental chemical over-exposure; case histories. **Ann Allergy 41**; 101-110 (1978)